A CASE OF DESTRUCTIVE LESIONS OF THE TEGMENTUM AND THALAMI OPTICI OF BOTH THE SUBTHALAMIC REGION OF THE LEFT, AND THE CORPUS STRIATUM AND NUCLEUS LENTIFORMIS OF THE RIGHT HEMISPHERES, WITHOUT APPARENT LOSS OF SENSATION OR MOTION.

By Dr. H. D. SCHMIDT,

PATHOLOGIST OF THE CHARITY HOSPITAL OF NEW ORLEANS.

Some time ago I met in the autopsy-room of the Charity Hospital a subject upon which, without my previous knowledge, an autopsy had been made by one of the resident students. When inquiring for the nature of the disease from which the patient had died, I learned from this student that the patient had not been an inmate of the wards in his charge, but that he had performed the post-mortem examination for one of his fellow students. All he knew of the case was, that the patient had, previous to his death, suffered from a slight diarrhœa, accompanied by an excessive debility; while his post-mortem examination had shown all the organs of the thorax and abdomen in a normal condition, excepting the small intestines, which had exhibited the signs of hyperæmia.

As the brain of this subject had remained untouched, I concluded to use this organ for some special anatomical purpose, and accordingly had it injected with a strong solution of chromic acid, before it was removed from the cranial cavity. But when examining it, after its removal from the cranium, I was surprised to find a part of the cerebellum—viz., the tonsils, pyramid, uvula, and nodolus—considerably softened, a condition which not only rendered the organ unfit for my special purpose, but, moreover, caused me to suspect the presence of some further lesions in its interior. I proceeded, therefore, to separate the cerebellum, medulla oblongata, and pons from the cerebrum by a section through the crura cerebri along the upper border of the pons, and just below the corpora quadrigemina, and found, as I had anticipated, an extensive destructive lesion in the parts thus exposed, involving the whole tegmentum and neighboring parts of the basis of the crura cerebri. A closer examination, however, revealed that the lesion extended to those parts of the cerebrum mentioned in the heading of this paper, and to be described hereafter.

Being forcibly struck by the want of correspondence existing between these extensive lesions of the brain and the clinical history of the case, as far as it had been told to me, I almost suspected some mistake to have been made, regarding the identity of the dead subject and the patient concerned in the history. But when directly interrogating the student in charge of the ward in which the patient had lived and died, I found that all I had thus far learned about this case was true, and that the physician of the ward himself had been unable to assign a proper immediate cause to the death of this patient.

The remarkable fact of the existence of such extensive destructive lesions, without any manifestation of a decided disturbance of sensation or motion on the part of the patient during life, as witnessed here, rendered the case in my eyes fully worthy of being reported. Through being at the time deeply engaged in the study of some other subjects, I must regret to have been unable to extend the microscopical examination to other parts of this brain than those mentioned; the results thus far obtained, however, are sufficiently correct to explain the nature of the lesions concerned.

The following are the more prominent points in the history of this case, furnished to me by the student of the ward, and endorsed by his physician:

Salvador, Charles, laborer, native of Italy, forty-two years of age, admitted to the Charity Hospital on January 19, 1885. Previous to his admittance he had resided six months in Plaguemine, La. His habits were irregular, and his hereditary history obscure. According to his statement, his general health had always been fair, with the exception of having been sunstruck two years ago, and having had swelling of the lower extremities while still living in Italy; otherwise he had never been ill. About six months previous to admittance to the Charity Hospital he had an attack of dysentery, from which he had more or less suffered until one month ago. Since that time he had felt a weakness and pain over the epigastric region. When admitted to the wards of the Hospital, on Jan. 19th the patient's general condition was poor, he was anæmic, and emaciated. His appetite was good, and his bowels were regular; there was no organ found to be enlarged, nor could any structural change be determined by physical examination. The only symptoms of disease observed were weakness and indefinite pain in the epigastric region. His pulse was full and strong—eighty-four beats in the minute; the respiration was twenty-four, and the temperature normal. The examination of the urine showed that this fluid contained a trace of albumin—about 1 per cent.,—a few leucocytes, and some shreds of epithelial cells from the mucous membrane of the bladder, while its spec. gr. was 1010. For this reason suspicions were entertained that the patient might suffer from an initiary attack of Bright's disease. Subsequent examinations of the urine showed that the albumin had disappeared, while the spec. gr. had remained the same. In addition to the above description of the patient's condition it remains to be mentioned that he suffered from inflammation of the cornea of the left eye, for which reason he went daily to the department of the Diseases of the Eye for treatment.

While, however, the condition of this patient remained from day to day, about the same as above stated, his emaciation and debility slowly increased until February 14th, when he was taken with a slight diarrhœa, though he had no fever, and his appetite remained good, while his pulse was strong and full. On February 18th, the patient was confined to his bed, and very weak, in which condition he died without an apparent cause.

I have been assured by both, the physician and the student of the ward, that they had not observed the slightest loss of sensation, or motion, or other nervous disturbances on the patient, the only striking phenomena had been the increasing emaciation and excessive debility.

After having stated the above meagre clinical history of the case, I shall now proceed to describe, as far as circumstances will allow, the details of the extent and nature of the destructive lesions, observed on the brain under consideration. At the same time, however, I must remind the reader that this description cannot but be to a certain extent incomplete, on account of the chromic-acid solution that had been injected into the vessels of the organ, and which, though not altering the histological character of the anatomical nervous elements to such a degree as to render them unfit for microscopical examination, nevertheless had obliterated the natural color of the respective white and gray substances, and, furthermore, had increased their natural consistence. As regards this point, however, I may say, that taking into account even the effect of this solution on the nervous tissues, the consistence of this brain appeared to me greater, and its bulk smaller, than I had previously observed on other brains injected by the same solution.

The principal lesions extended, as already mentioned before, from the fourth ventricle upwards, to the thalami optici of both hemispheres, and to the sub-thalamic region of the left one. From the fourth ventricle up to the crura cerebri, the tegmentum, enlarged in its dimensions, had preserved its consistence, while higher up it already appeared broken up into larger or smaller fragments of greater or lesser consistence. As far as I was able to judge this solution of continuity of structure did not exist during the life of the patient, but was rather caused by the force of the injection, which by rupturing some of the diseased blood-vessels, forced its way through the degenerated and softened nervous tissues, coagulating them, at the same time, in the form of larger and smaller fragments. The higher up the lesion extended the smaller and softer these fragments appeared, until in the thalamus opticus, nothing but the outer strata of the ganglion were left in the form of a shell. From this it appears that the degeneration had commenced in the cerebrum, and descended through the crura cerebri and pons downwards to the medulla oblongata. My observations show, furthermore, that in the two latter parts of the brain the disease remained principally confined to the tegmentum, while in the crura cerebri it was observed to have encroached, to a certain extent, upon the basis, though sufficient substance of the latter was left for the transmission of motor energy. The latter circumstance explains the absence of motor paralysis in the clinical history of the patient.

With the assistance of the accompanying drawings let us now consider the extent and nature of the disease in the pons Varolii and medulla oblongata, in which the degenerated nervous tissues had to a certain degree preserved their integrity. Fig. 1 represents a horizontal section of the two lateral halves of the pons Varolii, made at different heights, while Fig. 2 represents a sagittal section throughout the pons and medulla oblongata. In beginning with the horizontal section *a* on the left half of the pons, made about $\frac{1}{8}$ inch below the emergence of the crura cerebri, we observe the tegmentum *d* enlarged in its natural dimensions, and separated by a deep fissure, not only from the anterior part of the pons *c*, representing the pyramidal tract, or basis, of the respective crus cerebri, but also posteriorly from the lemniscus field *e* by a similar fissure; the course of the transverse nerve-fibres of the pons, as the drawing will show, has also been deranged by the increasing dimensions of the tegmentum. At the same time, a difference of shade is observed between the anterior and posterior part of the latter. The extent of the fissure which separated the tegmentum from the pyramidal tract may be seen in Fig. 2, *g*. In examining now the horizontal sections of the right half of the pons, made $\frac{1}{8}$ inch higher up, just below the emergence of the respective crus cerebri, as represented in Fig. 1, *b*, we observe the course of the transverse bundles of fibres of the pons still more deranged, while, beside the fissures already mentioned, another appears running through the middle of the tegmentum, and to separate the anterior light part of the latter from its posterior, or darker shaded portion of the pons. As on the left half, on the right half also, the lemniscus field is found entirely separated from

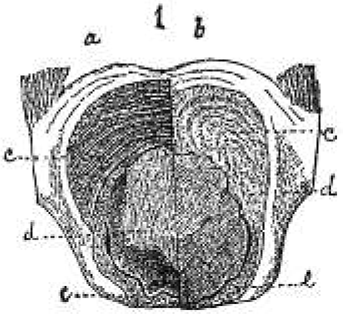

FIG. 1.

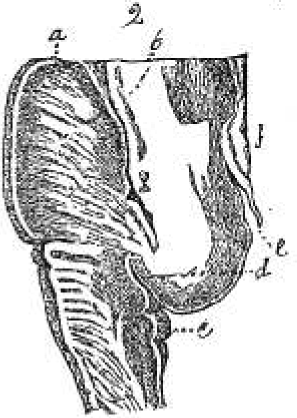

FIG. 2.

Explanation of the Illustrations.

Fig. 1.—Transverse section of pons Varolii ; *a*, one eighth of an inch below the emergence of the crura cerebri ; *b*, directly at the level with the emergence of the crura cerebri ; *c*, pyramidal tracts ; *d*, tegmentum ; *e*, leminscus field.

Fig. 2.—Sagittal section of pons Varolii and medulla oblongata ; *a*, pyramidal tract ; *b*, tegmentum, enlarged in its bulk, and projecting into the fourth ventricle ; *c*, entrance into the fourth ventricle (clava) ; *d*, fissure formed in the fourth ventricle by the projecting tegmentum ; *e*, aqueduct of Sylvius ; *f*, lingula ; *g*, fissure formed between the pyramidal tract and the tegmentum, described in the text.

the tegmentum. The examination of the sagittal section through the middle of the pons, as represented in Fig. 2, shows us the morbid downward growth of the tegmentum, bulging, in the form of a tumor, into the fourth ventricle. This enormous bulging into the latter gave rise, very probably, by the pressure which it produced, to the softening of those parts of the cerebellum mentioned before.

The higher up the morbid growth of the tegmentum extended, the more, as already stated before, it had undergone the softening process. On the left side, where the morbid growth extended throughout the whole tegmentum into the subthalamic region, and to the thalamus opticus of the left hemisphere, the softening process had been going on to the greatest extent, destroying the greater part of the latter, while also affecting the adjacent parts, such as the optic tract and the corpora geniculata, which were found considerably contracted. In the right hemisphere the same parts were invaded by the morbid growth, though not softened to the same extent as in the left.

Besides the lesions above described, there were a number of larger or smaller cavities found to have been formed in some parts of the left hemisphere. Thus, in the corpus striatum, a cavity 2 ctm. in length and height, and 1 ctm. wide, and communicating with smaller cavities, was met with in the nucleus lentiformis. Besides these, a number of smaller ones, about 1 mm. or more in diameter, were met with in both the corpus striatum and the nucleus lentiformis. Some of these smaller cavities were also found to exist in the cortex of the island of Reil and operculum. As regards the mode of formation of these cavities, we may presume that the larger ones were formed by the destruction of the walls, or septa, of a number of smaller ones, bordering one another. In the right hemisphere no cavities were found, but instead a number of prominences were observed upon the surface of the corpus striatum, which, when cut into, proved to be cavities, containing a serous fluid; they were probably formed in the same manner as the others—that is, by the breaking down of the neoplastic growth.

In examining carefully thin horizontal and longitudinal sections of the tegmentum of the pons it was found that the neoplastic element of the diseased parts, giving rise to their increased bulk, as well as inducing, at a later period, the process of softening, consisted of an exceedingly finely granular or fibrillary material, deposited between the nervous elements. But while many of the smaller blood-vessels of the diseased parts manifested a pathological condition by a considerable thickening of their adventitia, the ganglion cells and nerve-fibres showed no marked changes in their appearance. The only change to be noticed might have been the more than usual varicose form of the nerve-fibres. In some places the neoplastic element appeared entirely granular, whilst in others the minute granules had become arranged in rows, to form exceedingly fine fibrillæ, resembling those forming the neuroglia upon the surface of the cortex cerebri. The nuclei, or cells, belonging to the neuroglia of the diseased tegmentum appeared not to be increased in number. The walls of the various cavities in the corpus striatum and nucleus lentiformis presented the same structural changes as those above described.

Judging from this latter fact, I am inclined to regard the neoplastic material in this case as fibrinous in character, exuded from the blood-vessels as the product of a very slow inflammatory process.

Although there is nothing said in the history of this case which directly indicates the existence of some form of chronic cerebritis, the anatomical facts above stated nevertheless show that such a condition must have existed at one time or another; and the question might, therefore, be raised, whether the attack of sunstroke, from which the patient had suffered two years previous to his death, might not have been the exciting cause of a low chronic inflammation of the brain, giving ultimately rise to that morbid condition of this organ, described in the foregoing pages, and generally known as sclerosis of the brain. My own observations on the brains of sunstroke cases lead me to presume that such a condition existed in the brain of the above described case. Having, during the summer of 1865, taken in charge, for the purpose of special study, all the sunstruck cases that were brought to the Charity Hospital, I had opportunity to observe the condition of the brain of numerous fatal cases that occurred. In all these instances I found this organ highly congested, while, on the other hand, those patients who recovered from the attack underwent a very slow reconvalescence, suffering for a long time afterward from a deeply seated headache.